Pakistan Journal of Neurological Sciences (PJNS)

Volume 1 | Issue 2 Article 5

9-2006

Clinical Applications of Polysomnography

Imran I. Ali

Medical University of Ohio, Toledo, Ohio

CLINICAL APPLICATIONS OF POLYSOMNOGRAPHY

Imran I. Ali
Department of Neurology, Medical University of Ohio, Toledo, Ohio

Correspondence to: Imran I Ali, Professor of Neurology, Medical University of Ohio, Toledo, Ohio. E-mail: iali@meduohio.edu

Sleep is an important aspect of daily life for all of us. Disruption of a normal sleep wake cycle can lead to significant alteration of brain function. If prolonged or sustained this disruption can have significant social and medical consequences. In an era of increasing societal demands on individuals the amount of time available for sleep has been reduced significantly. The consequence of increasing sleep debt leads to reduced energy, fatigue, excessive daytime sleepiness and poor performance on tasks requiring attention and concentration. Interestingly, catastrophic accidents blamed on human error such as Exxon Valdez oil spill have most often occurred at times of greatest sleepiness and fatigue (which is usually between midnight and 8 am, as was the case with this disaster). Motor vehicle accidents are also more likely in patients who are sleep deprived with an extremely high mortality rate.[1] The impact of sleep deprivation on performance of health care workers has been well researched[2] and in the United States rules have been enacted by the accreditation bodies to limit the amount of time physician in training can work without sleep.

Sustained sleep deprivation also negatively impacts a number of different physiological functions including blood pressure, glucose tolerance and leptin levels.[3,4,5] Untreated sleep apnea is a well identified risk factor for myocardial infarction, hypertension and cerebrovascular disease which will be discussed in more detail later in this review.

CLINICAL APPROACH TO SLEEP DISORDERS

Approximately 10 % of the population has a sleep disorder making it one of the most common medical conditions seen in clinical practice. Insomnia is the most common sleep disorder, followed by sleep-disordered breathing (sleep apnea) and periodic limb movements of sleep. However, this review will concentrate on sleep apnea as the primary disorder of interest.

The prevalence of sleep-disordered breathing amongst adult men varies from 1-8 %, the rates being highest in those between 40-59 years of age.[6] The prevalence rates are much higher for men compared to women in this age group as well. The gender differences in young and the elderly do not appear to be as significant although men still outnumber women 2-3:1. The prevalence of obstructive sleep apnea is also higher in those with hypertension, diabetes, facial anomalies and obesity.

One of the interesting aspects of sleep disorders is that they may present with a seemingly unrelated medical problem. For example patients with obstructive sleep apnea syndrome (OSAS) may initially come to medical attention for hypertension, cerebral or myocardial infarction. Therefore a high index of suspicion is required to make the diagnosis. Risk factors associated with OSAS include male sex, age, obesity, habitual snoring, alcohol, opiate use, craniofacial abnormalities and endocrinopathies (such as hypothyroidism).

Common signs and symptoms associated with OSAS include snoring, excessive daytime sleepiness, early morning headaches and fatigue. Snoring may occur in approximately 30 % of the population but a substantial number of those individuals do not have OSAS. A complete neurological, cardiovascular and respiratory examination should be performed in each patient. Examination may show evidence of obesity and structural evidence for airway obstruction such as narrow nasal passage, long soft palate, large tonsils, or retroflexed mandible. These signs may not be seen in all patients with OSAS. In addition, there are many individuals with OSAS who are not obese. Clinical features have a sensitivity and specificity that is between 60-70% and are definitely inadequate to be used as the only tool for diagnosis of sleep disorders.

In addition to clinical testing screening questionnaires have been developed and validated for evaluation of sleep disorders. Epworth Sleepiness Scale (ESS)[7] is a useful measure of sleepiness and can be easily used in clinics to screen patients for excessive somnolence. The scores on this self administered test correlate well with respiratory disturbance index in patients with known obstructive sleep apnea but not with mean sleep latency test (MSLT). There are other screening questionnaires such as the Stanford Sleepiness Scale that can also be used in lieu of the ESS. In addition Sleep Disorder Questionnaire or the Berlin Questionnaire Inventory is more detailed and specific questionnaire that may be useful in evaluation of OSAS, but at the present time is used mainly as a research tool.

POLYSOMNOGRAPHY IN EVALUATION OF SLEEP RELATED DISORDERS

Currently, polysomnography is considered the gold standard for diagnosis of sleep disorders and practice parameters for polysomnography have recently been updated.[8] Indications for the study are listed in table 1.

Since the diagnosis is based on the results of this test, the study should be performed by an accredited laboratory with a well trained staff and qualified polysomnographer. The study may need to be repeated in patients who may have a "first night effect" or equivocal results. Portable or ambulatory studies have a very limited role in evaluation of sleep apnea and are mostly used in situations where patients cannot be studied in the laboratory.

TECHNICAL ASPECTS OF POLYSOMNOGRAM RECORDING PARAMETERS

An overnight polysomnogram is performed in a dedicated sleep laboratory with comfortable facilities that simulate an individual's home environment. The patient is expected to arrive 60-90 minutes prior to the start of the study. Standard electrodes are placed for recording EEG, EMG, eye movements (EOG), respiration, leg movement, oxygenation and EKG.

The standard EEG montage in a sleep study includes a central (C4) and occipital (O1) derivation. This allows identification of characteristic EEG changes such as the posterior dominant rhythm and sleep spindles which are characteristics of wakefulness and sleep, respectively. Additional set of electrodes can be placed if there is a concern for an associated seizure disorder. The recent development of digital systems has made this issue quite easy to resolve and most laboratories can now perform sleep studies with more elaborate EEG montages to improve the diagnostic capability. The paper speed is kept at a slower rate of 30 mm/second based on older analog system (which was done to conserve paper), however, since polysomnographers are used to that paper speed it is maintained as a standard even in digital recordings. Video recording is useful in conditions where the behavior needs to be correlated with sleep stage (such as in parosomnia) or with EEG (as in a seizure).

Two separate electrodes are placed 1 cm lateral and superior and 1 cm lateral and inferior to the to the lateral canthus for recording eye movements. These electrodes are usually referenced to the ipsilateral ear. Out of phase movements with this montage allows accurate identification of eye movements during REM sleep.

Electromyographic recording is performed with an electrode on the chin as a standard procedure. Chin EMG activity decreases with advancing sleep and is entirely absent during REM sleep. Additional electrodes can be placed on the anterior tibialis muscle bilaterally 3 cm apart. These electrodes are useful in identifying periodic limb movements during sleep.

Ventilatory monitoring is the most crucial aspect of a polysomnogram. Absence of ventilatory effort with cessation of airflow indicates a central apnea while ventilatory effort without airflow is consistent with obstructive sleep apnea. A mixed apnea is defined as an event that starts off as a central apnea but later develops a picture consistent with obstructive sleep apnea (that is ventilatory effort without airflow). Technically airflow can be monitored by thermistors, thermocouples, carbon dioxide detectors, nasal cannula or pneumotachography. The thermistors and thermocouples are the most commonly used of these instruments. Ventilatory effort can be monitored by esophageal pressure monitors, piezoelectric belts, impedance or inductive pneumography or strain gauges. Esophageal pressure monitors are extremely sensitive at measuring intrathoracic pressures but are of limited use except in equivocal cases because of the invasiveness of the procedure and the mild discomfort associated with their placement.

In addition oxygen saturation and electrocardiogram provide additional critical diagnostic information. In patients with OSAS significant oxygen desaturation may also be associated with cardiac arrhythmias which are considered a marker for the severity of apnea.

TABLE.1
Age Distribution of 100 patients with stroke

Standard	Optional
Diagnosis of Sleep Related Breathing Disorders (SRBD)	
Positive Airway Pressure titration in SRBD	Coronary Artery Disease*
Preoperative Evaluation of Patients with SRBD	TIA or stroke*
Follow up after medical or surgical treatment of SRBD	Parasomnia
After substantial (> 10 %) weight gain or loss on treatment	Nocturnal paroxysmal events (suspected seizures
Inadequate clinical response	or atypical parasomnias)
Congestive Heart Failure with symptoms of SRBD or	Restless Legs Syndrome
poor response to therapy	Insomnia
Neuromuscular disease with sleep related symptoms	Circadian Rhythm Disorders
Suspected narcolepsy	
Periodic Limb Movements of Sleep (PLMS)	

SLEEP SCORING

The method of scoring sleep is based on standard systems described previously 9. By convention sleep is scored in epochs of 30 seconds with changes that are sustained for greater than 50% of that particular epoch. The defined stages of wakefulness and sleep are as follows

Stage of Wakefulness: Defined as wake state with alpha frequencies noted posteriorly with mixed low voltage frequencies noted in the background EEG. Eye movements and muscle activity is also noted

Stage 1 Sleep: This is a state of sleep with low voltage activity with occasional vertex sharp waves and slow eye rolling movements. There is reduction in EMG activity with absence of sleep spindles and K complexes.

Stage 2 Sleep: Stage 2 requires the presence of sleep spindles and K complexes with less than 20 % of the epoch has delta activity.

Stage 3 Sleep: Stage 3 is defined as 2 Hz delta greater than 75 uv amplitude comprising 20-50 % of the record.

Stage 4 Sleep: Stage 4 is defined as 2 Hz delta activity of greater than 75 uv amplitude comprising > 50 % of an epoch.

REM sleep: Relatively low voltage mixed frequency with rapid eye movements and characteristic saw tooth waves. There is marked reduction in EMG with muscle atonia.

Useful definitions for interpretation of polysomnogram are listed in table 2.

TABLE.2
Definitions of terms used in PSG

Term	Definition
Awakening	Recurrence of alpha activity for >50 % of the epoch following a defined sleep state
Arousal	A shift to a faster EEG frequency for 3 or more seconds following a 10 second or greater period of a defined sleep state. During REM however, arousal is associated with change in EEG frequency and increased chin EMG activity.
Apnea	Complete or near complete cessation of breathing for > 10 seconds
Hypopnea	30 % reduction of thoracoabdominal effort or airflow for > 10 seconds with at least 4 % reduction in oxygen saturation
Apnea Index	The number of apnea episodes per hour of sleep
Hypopnea	The number of hypopnea episodes per hour of sleep
Apnea-Hypopnea Index (AHI)	The number of apnea and hypopnea episodes per hour of sleep
Respiratory Effort Related Arousal (RERA)	CNS arousals without meeting criteria for apnea or hypopnea
Respiratory Disturbance Index (RDI)	The number of apnea, hypopnea and RERA per hour of sleep

TEST RESULT INTERPRETATION

Prior to discussing abnormal sleep patterns it is important to recognize normal sleep physiology. Sleep onset begins with stage 1 sleep followed by stage 2 sleep. This is followed by stage 3 and 4 which are usually referred to as slow wave sleep. In most individuals there is a subsequent transition to stage 2 sleep followed by the first REM period which normally has a latency of 70-120 minutes. The subsequent periods of sleep have less slow wave sleep and greater REM sleep. The total number of cycles are about three to five in a full night's sleep. There is a decrease in amount of slow wave sleep and decrease in sleep efficiency as an individual gets older.

Defined abnormalities in polysomnograms may depend on clinical context in which they occur. Although there is limited clinical evidence more than 15 arousals per hour may be significant in a patient with excessive daytime sleepiness. Most often these arousals are associated with OSAS although may occur in other disease states such as periodic limb movements of sleep (PLMS).

The diagnostic criteria for OSAS (figure 1) include a minimum respiratory disturbance index (RDI) of 5 in symptomatic patients and greater than 15 in asymptomatic patients. Severity of OSAS is dictated by the number of apneic episodes per hour along with their duration and effect on oxygen saturation as well as presence of cardiac arrhythmias. Mild apnea is defined as RDI of less than 20, with 20-40 being considered moderate and greater than 40 considered severe apnea.[10]

Polysomnograms are also useful for assessment of other conditions such as periodic limb movements of sleep (PLMS). PLMS (figure 2) are defined as greater than 15 movements per hour. Each movement is scored when anterior tibialis EMG activity lasts 0.5-5 seconds with five movements occurring in a cluster with intervals of up to 90 seconds.

Other disorders such as insomnia, parasomnias and circadian rhythm disorders can also be studied providing useful information for the clinician. Some patients have difficulty falling asleep in a lab setting, the so called "first night effect" and may require a repeat study the next day. In patients with suspected narcolepsy it is important to obtain a routine polysomnogram prior to the mean sleep latency test to exclude sleep deprivation as a cause for REM intrusion and or hypersomnolence noted during the mean sleep latency test (MSLT). The MSLT is performed to assess daytime somnolence and sleep onset REM (SOREM). It involves four to five naps with standard EEG and chin EMG along with EKG recordings to assess different sleep states. The presence of SOREM and absolute sleep latency of less than 5 minutes is considered abnormal.

CEREBROVASCULAR DISEASE AND OBSTRUCTIVE SLEEP APNEA:

Cerebrovascular disease is one of the commonest neurological disorders with a high morbidity and mortality rate. Well-recognized risk factors for stroke include (but are not limited to) age, hypertension, diabetes, obesity, heart disease, hyperlipidemia, previous TIA or stroke and coronary artery disease. A frequently unrecognized but surprisingly common risk factor appears to be obstructive sleep apnea syndrome. In one study the PSG proven OSAS was five times more likely in patients with a history of stroke compared with controls matched for age, gender and body mass index. Interestingly in most patients the apnea appears to be obstructive rather than central and independent of the location of stroke. In the context of acute ischemic event one may see central apnea but usually resolves over time while the obstructive pattern persists suggesting that it is a pre-existing condition.[11]

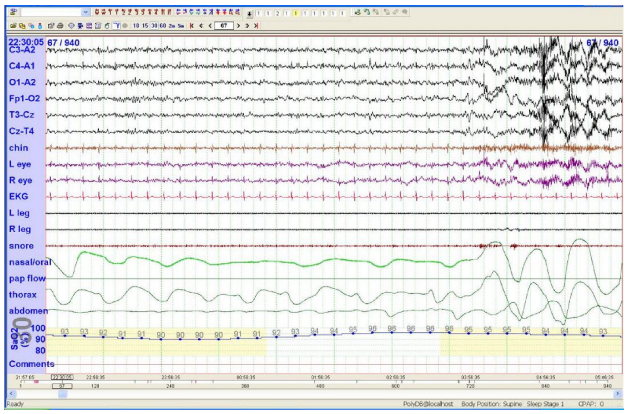

Figure 1: PSG showing classic obstructive sleep apnea associated with arousal (with permission and courtesy of Dr. Atif Hussein, Duke University).

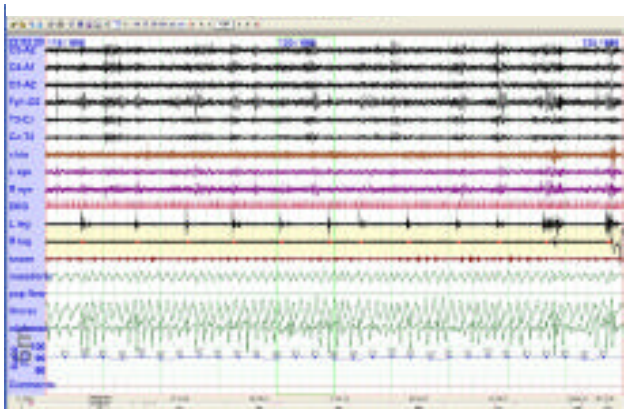

Figure 2: PSG showing periodic limb movements of sleep (with permission and courtesy of Dr. Atif Hussein, Duke University).

increased mortality, these include age, Apnea Hypopnea Index (AHI), presence of coronary artery disease and location of infract. Each unit increase in AHI increased mortality by approximately 5%12. Additionally appropriate intervention such as continuous positive airway pressure (CPAP) reduced the likelihood of recurrent stroke five fold.[13]

In summary early recognition and treatment of OSAS can reduce the incidence of stroke as well as positively impact on the morbidity and mortality associated with it. Clinicians involved in the care of patients with cerebrovascular disease should have a low threshold for evaluating these patients for undetected OSAS.

CONCLUSIONS:

Sleep related disorders are common and associated with significant morbidity and mortality. Polysomnography if done in a technically rigorous manner provides useful clinical information. Disorders such as OSAS if appropriately treated significantly improve patients' quality of life and reduce the morbidity and mortality associated with both cardiovascular and cerebrovascular disease.

REFERENCES

1. Pack AI, Pack AM, Rodgman E, et al. Characteristics of crashes attributed to the driver having fallen asleep. *Accid Anal Prec* 1995; **27**: 769-775
2. Landrigan CP, Rothschild JM, Cronin JW et al. Effect of reducing intern work hours on serious medical errors in intensive care units. *N Engl J Med* 2004; **351**: 1838-1848
3. Grigg-Damberger M. Why a polysomnogram should become part of diagnostic evaluation of stroke and TIA. *J Clin Neurophysiology* 2006; **23**: 21-38
4. Harsch IA, Konturek PC, Koebnick C, et al. Leptin and Ghrelin levels in patients with OSA: Effect of CPAP treatment. *Eur Respir J* 2003; **22**: 251-257
5. Carlson JT, Hedner ja, Ejnell H, et al. High prevalence of hypertension in sleep apnea patients independent of obesity. *Am J Respir Crit Care Med* 1994; **150**: 452-455
6. Cirignotta F, D' Alessandro R, Partinen M, et al. Prevalance of every night snoring and obstructive sleep apneas among 30-69 year old men in Bologna, Italy. *Acta Neurol Scand* 1989; **79**: 366-372
7. Johns MW. A new method for measuring daytime sleepiness: The Epworth Sleepiness Scale. *Sleep* 1991; **14**: 540-545
8. Kushida CA, Littner MR, Morgenthaler MD et al. Practice Parameters for the Indications for Polysomnogaphy and Related Procedures: An update for 2005. *Sleep* 2005; **28**: 499-519
9. Rechtschaffen A, Kales A, eds. A manual of standardized terminology, techniques and scoring for sleep stages of human subjects. Los Angeles: UCLA Brain Information Service/Brain Research Institute, 1968
10. Radke Ra. Sleep disorders: laboratory evaluation. In: Ebersole JS, Pedley TA eds. Current Practice of clinical electroencephalography, Third edition. William and Wilkins 2003: 803-822
11. Parra O, Arboix A, Montserrat JM, et al. Time course of sleep related breathing disorders in first ever stroke or TIA. *Am J Respir Crit Care Med* 2000; **1616**: 375-380
12. Parra O, Arboix A, Montserrat JM, et al. Sleep related breathing disorders: impact on mortality of cerebrovascular disease. *Eur J Respir J.* 2004; **24**: 267-272
13. Martinez Garcia MA, Galiano BR, Cabero SA. Continuous Positive Airway Pressure treatment in sleep apnea prevents new vascular events after ischemic stroke. *Chest* 2005; **128**: 2123-2129